

Acta Biotheoretica 47: 29-40, 1999

ON THE ADAPTIVE VALUE OF SOME MATE SELECTION STRATEGIES

Klaus Jaffe

Depto. Biología de Organismos
Universidad Simón Bolívar
Apartado 89000, Caracas 1080, Venezuela
Fax : 582-9063064/9063024
e-mail : kjaffe@usb.ve

Key words: Sexual selection, mate selection, gamete selection, evolution, ploidy, assortative mating

Running head: mate selection and evolution

ABSTRACT

Results of an agent-based computer simulation of the evolution of diploid sexual organisms showed that several mate selection strategies confer much higher average fitness to the simulated populations, and higher evolutionary stability to the alleles coding for these strategies, than random mating. Strategies which select for 'good genes' were very successful, and so were strategies based on assortative mating. The results support the hypothesis that mating is not likely to be random in nature and that the most successful mate selection strategies are those based on assortative mating or on advantageous genes.

INTRODUCTION

Although there is few evidence for mate selection in species reproducing sexually, there is no evidence for the absence of mate selection in any sexual species. That is, sexual reproduction without previous courtship behavior (or gamete selection) may be rare, as even yeast have been suggested to have mate choice (Nahon et al. 1995). Mate selection or courtship is commonly thought to assure that mates are of the same species and of different sex. However, most organisms have courtship behaviors which seem too elaborate to serve only this purpose. Thus, Darwin coined the concept of sexual selection, which has acquired a variety of meanings (Andersson 1994, Andersson and Iwasa 1996, Holland and Rice 1998, Murphy 1998). In addition to courtship, sexual reproduction may be associated with sophisticated gamete selection mechanisms. For example, studies of female choice (Eberhard 1996) suggest that sperm selection, as a complement to mate selection, is a rather generalized characteristic of organisms.

The effect of mate selection on biological evolution may be very complex and is not easily characterized with analytical models. Analytical models are limited in the degree of complexity they are able to tackle due to limitations in the mathematical tools available (Ruelle 1991, Cliff and Miller 1994, Levin et al. 1997). To avoid these limitations, evolutionary computer simulations have been developed for the study of the evolutionary dynamics (Hillis 1992, Kauffman and Johnson 1992, Miller and Todd, 1994, Jaffe 1996). Regarding sex, a few studies based on simulation models (Miller and Todd 1994, Davis 1995, Jaffe 1996, 1998), showed that mate selection affects biological evolution significantly. Theoretical studies of mate selection coupled to natural selection suggest that mate selection may reduce mutation load (Maynard Smith 1978, Taylor and Williams 1982, Heisler 1984, Kondrashov 1988), it may accelerate evolution by focusing natural selection to the appropriate genes (Jaffe 1996), it may be fundamental in explaining sympatric speciation (Todd and Miller 1991, and see references in Andersson 1994, and in Price 1998, for example), changing adaptive behavior (Miller and Todd 1994), and behavioral diversity (Werner and Todd 1997).

A problem in exploring the possible impact of mate selection on the evolutionary dynamics is the large amount of mate selection strategies possible. The strategy chosen will affect the results. Mate selection strategies aiming at reducing mutation loads showed a small improvement over random mating (Kondrashov, 1984, 1988), but assortative mating improved the fitness of population up to 4 times compared to those using random mating (Jaffe 1998). This improvement of assortative mating was evidenced only if the simulated organisms had more than 7 genes. Therefore, I used the model described in Jaffe (1998), simulating organisms with 14 genes, to explore the effect of various mate selection criteria on the evolutionary dynamics of sexual organisms, as this simulation model allows the study of features which emerge as a result of the interaction between mate selection and natural selection.

METHODS

The simulation model, described in detail elsewhere (Jaffe 1995, 1996, 1998), had better precision than analytical models in explaining experimental data regarding the emergence of genetic resistance to antibiotics and pesticides (Jaffe et al. 1997). In this agent based adaptive model, each individual was simulated as an autonomous agent which related to the environment and to other individuals according to the alleles it carried in its set of genes or loci as given in Table I. A population of such diploid agents, formed initially by a fixed number of individuals (i.e. agents), suffered each time step a five-step evolutionary process. Thus, a population of agents changed its genetic composition (frequency of alleles at the different loci) over time, due to differential mortality of the agents and due to the specific characteristics of the mating systems simulated. The five-step evolutionary process consisted of the following:

Mate selection: Females choose a mate so that mates were of the same species and of different sex, and according to mate selection criteria (MSC) defined by the allelic value of gene 8 (Table 1), as indicated in Table 2. The mate search was limited by the value of the allele of gene 13 (Table I). Females not finding the appropriate male mated with the one closest to the criteria searched for. If no conspecific male was found, the female did not reproduce during that time step.

Reproduction: Mated individuals produced offspring according to their phenotypically determined clutch size (see below), transmitting their genes following Mendelian rules for bisexual diploid reproduction: each parent provided half of the genes to the newborn, which then had two alleles for each gene respectively. One allele in the offspring came from the father and the other from the mother, determined randomly.

Variation: Randomly selected genes mutated randomly, changing their allelic value with a genetically determined probability (gene 10 in Table I).

Phenotypic expression: Mendelian allele inactivation was simulated. That is, only one randomly selected allele per diploid loci was expressed phenotypically. Three life history traits, similar to what is known to be common among animals (Roff 1992), were simulated. These traits were partially determined by three genes: Maximal possible life span, maximal possible clutch size and age for starting reproduction (genes 1-3 in Table I). That is, the final clutch size of each individual was calculated based on the allelic characteristic of the gene coding for clutch size and the age of the reproducing individual, using a normal distribution, so that :

$$cs(i) = M(i,cs,l) / [(oar^{0.5})^{-\{[(age(i)-oar)^2] / oar\}}] \quad \dots (1)$$

where $cs(i)$ is the clutch size of individual i , $M(i,cs,1)$ is the allelic value for maximum clutch size in the phenotypically expressed genome of individual i , $age(i)$ is the age of individual i , oar is optimal age for reproduction fixed as an external parameter in each simulation. That is, the

maximal clutch sizes were partially predetermined genetically and occurred at an optimal age of reproduction. Clutch size affected the probabilities of survival of the future adult, so that individuals born in clutches larger than optimal decreased their probability of survival exponentially. The equation here was

$$\text{if } cs(i) \leq ocs \text{ then } fit(k) = 1 \quad \text{else } fit(k) = ocs/cs(i)^3 \quad \dots (2)$$

where $cs(i)$ is the size of the clutch in which individual k was born, ocs is optimal clutch size fixed as an external parameter in each simulation, and $1/fit(k)$ is the additional probability for the offspring k being eliminated by random selection (see below).

Selection: The model did not assume any simplified expression of fitness but reproduction and individual survival were decomposed into different aspects for selection to act. Individuals were excluded from the population when any of the following criteria applied:

- 1- Their age exceeded their genetically prefixed life span.
- 2- When randomly selected with a probability which increased with population density and with the value $1/fit(i)$ in equation 2.
- 3- When parents clutch size exceeded optimal sizes at high population densities.
- 4- When a biocide was applied to randomly trim the population from individuals not possessing the right resistant phenotype: Individuals with non resistant phenotypes of genes 6 and 7 in Table I were killed randomly with probabilities which varied randomly from 0 to 0.95.

Two different type of experiments were performed in which organisms had to compete for limited computer memory and time in a variable environment.

- 1- Survival probabilities of **Isolated populations** were assessed by randomly creating initial populations with uniform distribution of alleles, in which all individuals had the same mate selection strategy. Then, the probabilities of these populations to survive a variable environment a given number of time steps were evaluated.
- 2- Simulations of **Gene selection** were aimed at assessing the effect of selection on the allelic distribution in gene 8, in a single interbreeding population. Different alleles for the various genes were assigned to each individual initially uniformly at random, and the temporal variations of the frequency in the population, of the various alleles coding for specific mate selection strategies, was monitored.

Other parameters were chosen based on previous results optimizing mate selection (Jaffe 1998). These were: optimal size of the population (or size above which density dependent selection increased exponentially = 800), initial size of the population (= 500), optimal clutch sizes (or clutch size above which fitness of offspring is reduced = 4), optimal age for reproduction (or age before and after which reproduction will decrease the fitness of mother and offspring = 2 time steps). Genetic complexity of simulated organisms (14 genes as given in Table I).

RESULTS AND DISCUSSION

The effect of each of these mate selection criteria on evolution in diploid organisms is shown in Figures 1 and 2. Figure 1 shows the results of the evolution experiments simulating isolated populations, each with a given MSC as indicated in Table II, whereas Figure 2 presents the results of simulations of gene selection. These results may be grouped into three categories:

1- Trivial but important results: All three MSC in which females chose males according to beneficial genetic traits (MSC 3, 4 and 5) outperformed random mating when these strategies were tested in isolated populations. That is, the more good genes are selected through mate selection, the greater the average fitness of the population (Fig. 1) and the more probable that the alleles coding for these MSC are maintained in a population (Fig. 2). These results suggest that selection criteria which somehow increase the odds of mating with a fitter mate are very successful. Several examples of secondary sexual characters as fitness indicators in real organisms are known (Eberhard 1985, Andersson 1994, for example). The model predicts that the more genes are associated to a sexual signal, the more likely that it is selected for mate selection. This seems to be the case, for example, of bilateral symmetry as a cue for mate preference (Pennisi 1995), where several genes affect directly or indirectly the degree of symmetry of the organism, which in turn determines the odds to be selected by a mate.

2- Surprising results: Pure sexual selection (MSC 1) in which females chose males with strong sexual signals or sex appeal not directly related to fitness, induced rapid fixation of the allele producing the highest sex appeal (in less than 15 time steps in 95 % of 200 simulations). Populations with this mate selection strategy had a slightly higher but statistically significant survival probability compared to random mating (Fig. 1, chi-squared test between MSC 0 and MSC 1: $p < 0.01$, 500 simulations). In addition, when gene selection was simulated, a slight but continuous increase over time in the frequency of alleles coding for this strategy was observed (Fig 2b). These results help validate the model as they show that sexual selection increases mating success of the emitter, and thus increase its fitness. The results also show that the simulations produce run-away sexual selection (Houde and Endler 1990, Bakker 1993, Houde 1994) in that stronger sexual signals (i.e. sex appeal) are fixed very fast by the evolutionary dynamics. The increase in the fitness of populations formed by individuals which had only the MSC 1 allele (Fig. 1) has to be explained due to genetic drift (Soler et al. 1996, Jaffe 1996), as the signal for sex appeal may frequently be associated to the presence of good genes (at least in these simulations), being then a good predictor of fitness. The simulation will then select organisms with both, good genes and the appropriate signal for sex appeal, even if the

algorithm used in the simulation does not explicitly relate sex appeal with any additional trait conferring higher fitness. An indirect confirmation of these results is that recent evidence suggest that sexual signals which somehow increase mating success induce divergence of the trait mainly due to sexual selection (Arnqvist 1998), showing that sexual selection may fix genetic traits faster than natural selection.

The handicap simulation (MSC 2), consistently failed to improve on random mating (MSC 0). The adaptive disadvantage of the simulated handicap seemed to be too strong, and thus, populations in which females selected males based on this handicap had low survival probabilities (Fig 1), and alleles coding for this MSC were displaced by other alleles in interbreeding populations (Fig 2). This result could also be due to the fact that a high initial handicap does not allow the coupling of advantageous genes with the signal for handicap. The results suggest that sexual selection based on a handicap is likely only if the handicap is initially very small so that the signal may be coupled to good genes by the evolutionary dynamics (see also Andersson and Iwasa 1996, for example).

The fact that selection of old males (MSC 7) was less adaptive than that of young males (MSC 6), was unexpected (Fig. 1, $p < 0.005$, chi-squared test, $n = 200$). It might be thought that old males had shown they ability to survive and thus age should be an acceptable indicator for fitness. These simulations tell us otherwise. A tentative explanation here is that young males have recently been produced by the fittest parents and thus had a better genetic composition than older ones. The relative advantage of mating with old or young males may depend on the specific relation between live span and time steps used in the simulation and was not explored further.

3- Assortative mating: An interesting result was found with simulations of assortative mating, confirming its importance in biological evolution (Davis 1995, Jaffe 1998). Although active assortative mating was simulated, the results may also relate to passive assorting. That is, individuals may chose genetically similar reproductive mates actively or external constraints may force low dispersal rates, imposing mating among similar mates (high inbreeding). In both cases the result is that mating occurs among genetically similar individuals. Alleles coding for assortative msc (MSC 10 and 11 in Table 2) dominated in simulations of gene selection (Fig 2), but when measuring the survival probability of isolated populations (Fig 1) it was assortative mating based on genes different to that coding for msc (MSC 8 and 11) that conferred relatively high survival probabilities to populations. Assortative mating based only on msc (MSC 10), in which females chose males with their same MSC, was particularly successful in simulations of gene selection (Fig. 2), but in simulations of isolated populations it was not better than random mating (Fig. 1, $p > 0.1$, chi-squared test, $n = 200$). That is, alleles for assortative msc had a strong penetrating power, displacing other alleles in simulations of gene selection, but did not improve survival probabilities relative to random mating. The advantage

of assortative mating in simulations of gene selection is based on the fact that females select males which have their alleles for assortative mating, increasing the frequency of these alleles relative to other alleles, making it a strategy with high evolutionary stability in terms of the genetic dynamics, but with little effect on the average fitness of populations. Otherwise, assortative mating based on more general criteria (MSC 8 and MSC 11, Fig. 1), had a comparable fitness to MSC in which selection criteria were directly linked to advantageous genes (MSC 4 for example). Although it is not completely clear how assortative mating displaces other strategies in the simulations, several reasons (or a combination of them) may explain this: Assortative mating may reduce excessive allelic variability caused by sexual reproduction, especially among genetically complex organisms. It may allow fit females to maintain (not diluting) their advantageous genetic composition during reproduction, providing their offspring with a similar advantageous genetic outfit. It may accelerate the extinction of sub-optimal genetic combinations, as contrary to strategies selecting for good genes, assortative mating induces suboptimal females to mate with suboptimal males, thus accelerating the extinction of suboptimal alleles. The results showed that strategies based on assortative mating are the most likely to be selected through the evolutionary dynamics (i.e. gene selection, Fig 2), and thus should be common among real diploid organisms. Few cases have been thoroughly explored, although some controversial evidence for assortative mating among humans exist (Thiessen and Gregg 1980, Buss 1989, Rushton 1989, Grammer 1989, Voland and Engel 1990, Jaffe and Chacon 1995, but see the open peer commentaries in Rushton 1989 for example).

Selection by females of genetically different males increased allelic variability too much, making this strategy evolutionary unstable compared to random mating. The fact that dissortative mating was evolutionary unsuccessful is contrary to some findings in nature. For example, some organisms select their mates so as to increase the variability of the immune systems (Weedekind et al. 1995), by preferring mates with a different Major Histocompatibility Complex. This contradiction may be explained by the fact that in the present model, optimal allelic configurations existed. In simulations of the Red Queen hypothesis, in which co-evolution between parasites and host is simulated, dissortative mating based on genes conferring resistance to parasites showed to be more successful than assortative mating (Ochoa and Jaffe 1997).

The intensity of mate selection affected the success of the mate selection strategy. In the case of assortative mating (Fig 3), the screening of a maximum of five mates was sufficient to show a significant advantage over random mating. Excessive screening did not seem to improve the efficiency of mate selection, but rather tended to diminish it, although slower so in large populations.

CONCLUSIONS

The main result of the simulations is that mate selection is not likely to be random in nature. Several mate selection strategies (including strategies not explored here) may confer higher fitness and have a higher evolutionary stability than random mating. Thus, real organisms are very likely to use one or more mate selection criteria instead of mating randomly. Mate selection criteria, in order to increase adaptation, have to screen a large number of genes. Thus, phenotypes, affected by a large number of genes are predicted to be very efficient as mate selection signals, as seems to be the case for bilateral symmetry (Moeller and Thornhill 1998). The present work predicts that a certain degree of assortative mating should be favored by evolution, and thus, should be common among real organisms. Clearly, data on mate selection criteria from plant and animal studies are insufficient to reach a strong conclusion on the subject, but this theoretical exercise may stimulate investigations in this direction.

The simulation model did not considered gamete selection (i.e. sperm competition and sperm selection, etc). Several of the mate selection strategies outlined here my work at the level of the interaction between gametes. Therefore, any practical prediction derived from this work should assess the outcome of the reproductive process, i.e. should study also the offspring rather than focus on mating alone. Two specific prediction derived from this work, which might be tested experimentally, are:

1- Assortative mating should be common in diploid and polyploid plants and animals. For example, pollination strategies that maximize outbreeding should be rare and where they exist plants should have poor seed dispersal mechanisms, making it likely that pollination occurs between neighboring plants which are genetically related.

2- Mate selection mechanisms in plant and animals are expected to be quite sophisticated in that individuals should select mates based on several criteria which reflect good genes and/or similar genetic composition. Excessive genetic similarity between mates is also to be avoided, so that for each species and population an optimal degree of genetic similarity between successful mates (i.e. gametes producing the offspring) should be expected.

Acknowledgments : Thanks are due to G. Ochoa, C. Bosque, E. Herrera, N. Blüthgen and an anonymous referee for critical comments.

REFERENCES:

Andersson M.B. 1994. *Sexual Selection*. Princeton University Press.

Andersson M.B. and Y. Iwasa, 1996. Sexual selection. *Trends in Evolutionary Ecology*, 11 : 53-58.

Arnqvist G. 1998. Comparative evidence for the evolution of genitalia by sexual selection. *Nature* 393: 784-786.

Bakker T.C.M. 1993. Positive genetic correlation between female preference and preferred male ornaments in sticklebacks. *Nature* 363: 255-257.

Buss D. 1989. Sex differences in human mate preferences: Evolutionary hypotheses tested in 37 cultures. *Behavioral and Brain Sciences*, 12: 1-49.

Cliff D. and G.F. Miller, 1994. Protean behavior in dynamic games : arguments for the co-evolution of pursuit-avoidance tactics. In : Cliff P., P. Husbands, A Meyer and S. Wilson (Editors). *Proceedings of the Third International Conference on Simulation of Adaptive Behavior (SAB 94)*, M.I.T. Press, Bradford Books.

Davis, C.H. 1995. The effect of assortative mating and environmental variation on selection for sexual reproduction. *Evolutionary Theory* 11 : 51-53.

Eberhard W.G. 1985. *Sexual selection and animal genitalia*. Harvard Univ. Press, Cambridge, Massachusetts 243 pp.

Eberhard W.G. 1996. *Female Control : Sexual Selection by Cryptic Female Choice*. Princeton University Press, New Jersey, USA. 501 pp.

Grammer K. 1989. Human courtship behaviour: biological basis and cognitive processing. In: Rasa A.E., Vogel C. and Volland E. (Editors). *The sociobiology of sexual and reproductive strategies* Chapman and Hall, London, pp. 147-169.

Heisler, I.L. 1984. A quantitative genetic model for the origin of mating preferences. *Evolution* 36: 1283-

Hillis W.D. 1992. Co-evolving parasites improve simulated evolution as an optimization

procedure. In : Artificial Life II, SFI Studies in the Science of Complexity. C. Langton, C. Taylor, J.D. Farmer and S. Rasmussen, Editors. AddisonWestley Publishing Company.

Holland B. and W.R. Rice 1998. Perspective - chase-away sexual selection - antagonistic seduction versus resistance. *Evolution* 52: 1-7.

Houde A.E. 1994. Effect of artificial selection on male colour pattern and mating preferences of female guppies. *Proc. R. Soc. London B.* 256: 125-130.

Houde A.E. and J.A. Endler 1990. Correlated evolution of female mating preferences and male color patterns in the guppy *Pecilia reticulata*. *Science* 248: 1405-1408.

Jaffe K. 1995. Biodynamica: A simulation -model for biological evolution. File Biodynin.exe in <http://hercules.usb.vt/~kjaffe> (requires a MS Windows environment).

Jaffe K. 1996. The dynamics of the evolution of sex: Why the sexes are, in fact, always two? *Interciencia* 21: 259-267 and *errata* in 22: 48.

Jaffe, K. 1998. Sex, male selection and evolution. Proc. 7th Inter. Conference of Evolutionary Programming, San Diego, California, USA.

Jaffe K. and G. Chacon-Puignau, 1995. Assortative mating: sex differences in mate selection for married and unmarried couples. *Human Biology*, 67: 111-120.

Jaffe K., S. Issa, E. Daniels and D. Haile, 1997. Dynamics of the emergence of genetic resistance to pesticides among asexual and sexual organisms. *Journal of Theoretical Biology* 188: 289-299.

Kauffman S.A. and S. Jonson, 1992. Co-evolution to the edge of chaos : Coupled fitness landscapes, poised states and coevolutionary avalanches. In : Artificial Life II, SFI Studies in the Science of Complexity. C. Langton, C. Taylor, J.D. Farmer and S. Rasmussen, Editors. Addison-Westley Publishing Company.

Kondrashov, A.S. 1984. Deleterious mutations as an evolutionary factor. I. the advantage of recombinations. *Genet. Res.* 44: 199-.

Kondrashov, A.S. 1988. Deleterious mutations as an evolutionary factor. III. Mating preferences and some general remarks. *J. Theor. Biol.* 131: 487-496.

Levin S.A., B. Grenfell, A. Hastings and A.S. Perelson, 1997. Mathematical and computational challenges in population biology and ecosystem science. *Science*, 275 : 334-343.

Maynard-Smith i.M. 1978. *The Evolution of Sex*. Cambridge University Press, U.K.

Miller G.F. and P.M. Todd, 1994. Evolutionary wanderlust: Sexual selection with directional mate preferences. In : D. Cliff, P. Husbands, J.A. Meyer and S. Wilson Editors. *Proceedings of the Third International Conference on Simulation of Adaptive Behavior (SAB-94)*, M.I.T. Press, Bradford Books, pp. 21-30.

Moeller A.P. and Thornhill R. 1998. Bilateral symmetry and sexual selection - a metaanalysis. *American Naturalist* 151: 174-192.

Murphy C.G. 1998. Interaction-independent sexual selection and the mechanisms of sexual selection. *Evolution* 52: 8-18.

Nahon, E., D. Atzmony, A. Zahavi and D. Granot, 1995. Mate selection in yeast : a reconsideration of the signal and the message encoded by them. *Journal of Theoretical Biology*, 172 : 315-322.

Ochoa G. and K. Jaffe 1997. On sex, parasites and the red queen. Forth European Congress on Artificial Life, Sussex, UK.

Pennisi E. 1995. Imperfect Match, do ideal mates come in symmetrical packages ? *Science News*, 147 : 60-61.

Price T. 1998. Sexual selection and natural selection in bird speciation. *Phil. Trans. Royal Soc. London, Series B*, 353: 251-260.

Roff D.A. 1992. *The Evolution of Life Histories*. Chapman & Hall, NY, 535 pp.

Ruelle D. 1991. *Chance and Chaos*. Princeton University Press, Princeton, 195 pp.

Rushton J.P. 1989. Genetic similarity, human altruism and group selection. *Behavioral and Brain Sciences*, 12 : 503-559.

Soler R.V., J. Bascompte and S.C. Manrubia, 1996. Extinction : bad genes or weak chaos?

Proceedings of the Royal Society of London B, 263 : 1407-1413.

Taylor, P.D. and Williams, G.C. 1982. The lek paradox is not resolved. *Theor. Pop. Biol.* 22: 392- .

Thiessen D. and B. Gregg, 1980. Human assortative mating and genetic equilibrium: an evolutionary perspective. *Ethology and Socobiology*, 1: 111-140.

Todd P.M. and G.F. Miller, 1991. On the sympatric origin of species : Mercurial mating in the Quicksilver model. In R.K. Belew and L.B. Booker (eds.), *Proc. IV Intern. Conf. on Genetic Algorithms* (pp 547-554). San Mateo, CA : Morgan Kaufmann

Voland E. and C. Engel, 1990. Female choice in humans: a conditional mate selection strategy of the Krummhorn women (Germany, 1720-1874). *Ethology*, 84: 144-154.

Wedekind C., T. Seebeck, F. Bettens and J. Paepke, 1995. MHC-dependent mate preferences in humans. *Proceedings of the Royal Society of London B*, 260: 245-249.

Werner G.M. and P.M. Todd, 1997. Too many love songs sexual selection and the evolution of communication. In *Forth European Conference on Artificial Life*, P. Husbands and I. Harvey (eds.). MIT Press, Cambridge, Massachusetts.

Table 1: Genes and their possible alleles defining the organisms simulated. Organism had alle variance in all 14 genes, except when simulating isolated populations where all organisms had same allele for msc.

| Gene | Alleles | Effect on phenotype |
|------|---------|---|
| 1 | 0-10 | Maximum life span coding for life spans from 0 to 10 time steps. |
| 2 | 0-10 | Maximal clutch size. |
| 3 | 0-5 | Minimum age for initiating reproduction of females in t-steps. |
| 4 | 1-2 | Sex, either male or female. |
| 5 | 0-10 | Sex appeal of males towards females in an increasing range of attractiveness. |
| 6 | 0-10 | Resistance to biocide 1 . Only allele 0 was resistant to that particular biocide. |
| 7 | 0-10 | Resistance to biocide 2 as for gene 6. |
| 8 | 0-11 | MSC or criteria for mate selection (Table 2). |
| 9 | 0-5 | Minimum age for initiating reproduction of males . |
| 10 | 0-10 | Mutation probability coding for mutation rates from 0.2 to 10^7 random mutations per gene with logarithmic increments. |
| 11 | 0-1 | Sex determination mechanism, either random or weighted according to value of gene 12. |
| 12 | 0-10 | Sex ratio of newborns. |
| 13 | 1-100 | Number of males screened by females in search of potential mates before mating. |
| 14 | 0-10 | Neutral genes whose phenotypic expression has no effect on fitness. |

Table 2: Algorithms coded by the alleles of gene 8 which is responsible for the mate selection criteria (MSC) used by females in selecting conspecific males.

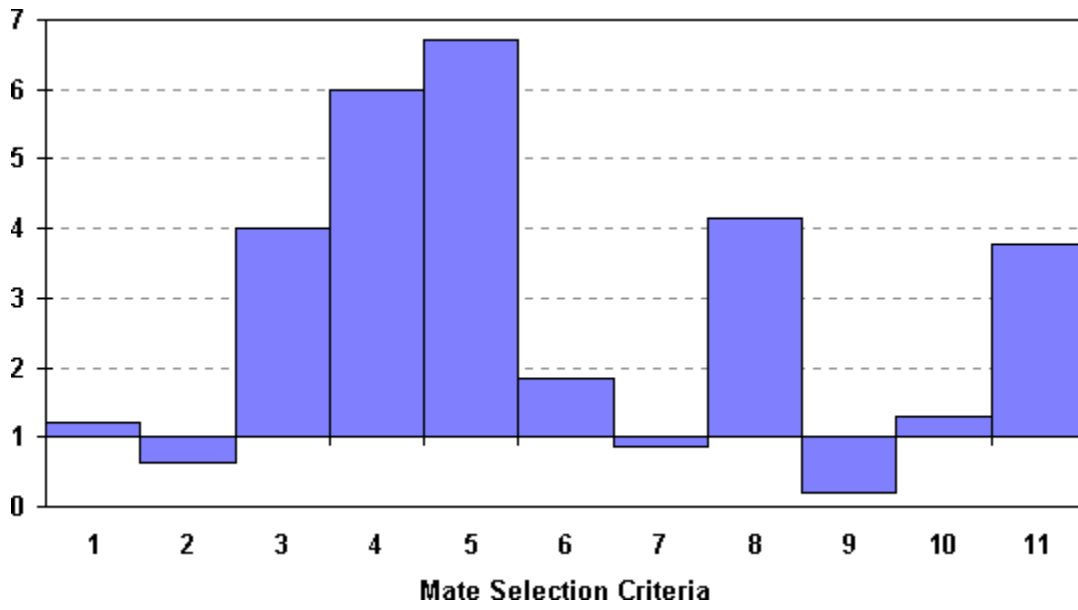
MSC Mate selection strategy coded

- 0 **Random mating:** females mate with the first conspecific male encountered.
- 1 **Neutral sexual selection:** Females prefer males showing alleles with high values in their gene for sex appeal (gene 5 in Table I).
- 2 **Handicap:** Females prefer males with high values in their resistance gene 6, i.e. they prefer males susceptible to the biocide, which if alive is because they might (or might not) be better in other aspects which compensate for this susceptibility.
- 3 **1 Good gene:** Females prefer males with low values in gene 6, i.e., preferred resistant males, i.e., selection of one 'good gene'.
- 4 **2 Good genes:** Females prefer resistant males for both biocides, i.e., selection of two 'good genes'.
- 5 **5 Good genes:** Females prefer males with appropriate allelic values in genes for maximum life span, clutch size, age for females to start reproducing, and the two resistance genes (genes 1,2,3,6 and 7), i.e., selection of 5 'good genes'.
- 6 **Youth:** Females prefer young males.
- 7 **Experience:** Females prefer old males.
- 8 **Assortative mating - msc:** Females screen the allelic values of males and chose those with an allelic composition most similar to hers. All genes are screened, except gene 8 coding for MSC.
- 9 **Dissortative mating:** As MSC 8 but females prefer males with the most dissimilar alleles.
- 10 **Assortative msc:** Females prefer males which have her allele for *MSC*.
- 11 **Assortative mating:** Females prefer males with a similar allelic composition, screening all genes, including the gene for MSC (MSC 11 = MSC 10 + MSC 8).

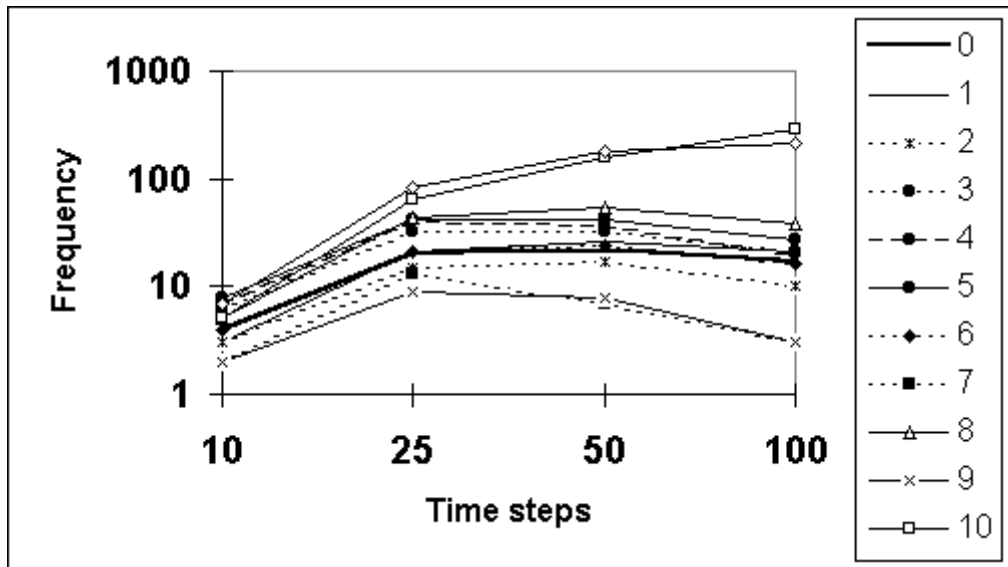
Figure 1: Probability of survival of 'isolated populations' in which all individuals are diploid and use an single mate selection criterion. Probabilities are expressed as a multiple of the probability of survival of populations using random mating. Mate selection criteria used are indicated with numbers in the ordinate, and correspond to the MSC given Table 2. For each MSC, a minimum of 200 simulations were run for 30 time steps. Gene 8 (MSC) had a fixed allelic value in each simulation, as indicated in the ordinate, corresponding to the alleles defined in Table 2.

Figure 2: Average number of alleles in simulations of 'Gene selection' in which individuals coded their mate selection strategy according to alleles of gene 8 which has allelic variance. The y-axis gives the average among simulations of the absolute number of each allele present in the population at different time steps. Numbers indicated in the legend correspond to allelic values in Table 2. Else as in Figure 1. Fig. 2a includes strategies with assortative mating whereas Fig. 2b excludes them.

Figure 3: Effect of the mate selection intensity (number of males screened before mating) on the probability of survival of isolated populations of organisms using assortative mating. Values on the right y-axis indicated survival probabilities when the number of males screened was variable and was defined by alleles in gene 13, also subjected to adaptation. Curves indicated with squares, circles and triangles are for simulations with optimal size of the population values of 200, 400 and 800 individuals respectively.



a



b

